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Post-traumatic stress disorder and stuttering: a diagnostic challenge in a case study

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Abstract

Mr. B., a 57- year- old three multilingual executive in an American Company was victim of a road accident. He was brought to hospital where no obvious lesions were found. Following this accident, he suffered from constant nightmares with flashbacks of the accident, insomnia, loss of concentration, loss of memory and attention, emotional instability, great fatigue and anxiety. As to the speech problems, he suffered from word finding problems and stuttering. All the specialist reports (psychiatrist, first RMI etc.) concluded that he suffered from Post Traumatic Stress Disorder. When he came to see me for a neurolinguistic analysis of his stuttering, I was puzzled by different aspects of his speech that did not fit in with "psychogenic stuttering following a shock". The analysis of his speech and the speech problems opened the way to more investigations. This case study is emblematic for/of the assumption that very careful neurolinguistic analysis can detect neurological dysfunction linked to fine, discrete and diffuse lesions that might not be immediately detected by fRMI.

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Keywords: PTSD; neurogenic; stuttering; fRMI

1. Introduction

Post-Traumatic Stress Disorder (PTSD) is well documented and defined as "a natural emotional reaction to a deeply shocking major life-threatening event. It can result from several types of emotionally shocking experience" (Diagnostic and Statistical Manual of the American Psychiatric Association)

The generally accepted criteria for PTSD are the following:

1. Experience of a traumatic, life-threatening event,
2. That is persistently re-experienced, with:

A. Intrusive psychological distress related to it, and

B. Physiological reactions to several aspects of the event.

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The observed Symptoms of PTSD are:

- Intense fear, even horror, helplessness, sleep disturbances (that give rise to chronic fatigue) , irritability, concentration problems, feelings of detachment, impaired memory, learning difficulties and emotional disturbances.
- If the symptoms extend beyond some months, they can reflect a persistent, abnormal adaptation of neurobiological systems to stress, at the level of endocrine and neurotransmitter pathways and brain regions that regulate fear. The well-known risk factors for PTSD are:
- A history of stress ante-accident and increased cortisol response to stress, low years of education, prior psychiatric history, child abuse and lack of social support (Bremner, 2003, 2008)
- The clinical observation shows that PTSD is often accompanied by devastating functional impairment.

2. The patient

The patient is a 57 year-old, high-educated man who was a victim of a severe car accident on his way home from his work. He had no history of stuttering ante-trauma. He had no loss of consciousness after the accident, but was very shocked, because of the lack of help. As a matter of fact the ambulance couldn't get through the traffic jam due to the general terrible road conditions (the traffic net was paralyzed by generalized ice). The patient couldn't drive. He was brought home by another driver.

The day post-accident he wasn't very well and went to the hospital to undergo some investigations. The Scan showed no traumatic peculiarities, but neuropsychological observation revealed some striking elements, such as deficits in memory and concentration, very high anxiety arousal, nightmares in relation to the accident, some hesitations during speech, and the most striking fact of all, the patient manifested very, very slow speech, which was completely new for him. He got some days of rest to help him overcome these deficits.

However, the difficulties worsened during the following months and Stuttering was clearly manifest after 3-4 weeks. He then underwent again psychiatric and neuropsychological investigations that concluded with the diagnosis of PTSD and Stuttering.

As the car accident took place on his way home from work, a series of medical investigations had to be undertaken to decide whether his disabilities were due to the accident or to an other pre-existing neurological/psychiatric deficit or illness (important for the insurance company).

A month post-trauma those medical investigations took place, which confirmed the stuttering and word-finding problems. The very slow speech was still present. The patient's complaints were, besides the speech problems, constant nightmares (flashbacks of the accident), but no phobia, and an important loss of libido compared to his emotional life before the accident. In general, he noted changes in his emotional reactions which were sometimes exaggerated and sometimes abnormally flawed.

Neurological examination at that time revealed a loss of sensibility in the right leg and arm, also in the right part of the face. He was advised to take Seroxat, Mirtazapin and Remergon as medication. He was then left alone, tried to get back to his work, but couldn't function anymore. He was unable to start working, to write a mail in due time and without grammatical errors. He forgot words in his mails and was unable to detect them afterwards. He noted that he forgot elementary things to do: he forgot to close the door when he went out, to close the door of the fridge after opening, to answer a phone call etc. The situation didn't get better. As a matter of fact, it got worse.

3. Neurolinguistic examination

6 months later neurolinguistic examination revealed an aggravation of the stuttering symptoms such as:

- Repetitions and blocks with tension at the beginning of words/syllables
- Many abnormal pauses between phrases and within phrases and words
- After a pause, he often didn't know anymore what he was going to say
- The patient was very conscious of the very slow speech that irritated him. During communication the patient kept eye contact with his communication partner. He didn't manifest interjections or starters in his speech. No change of pitch and no so-called secondary reactions were observed.

Neurolinguistic Testing included the following:

- Counting 1-10, 1-20: revealed the loss of automatic series: many hesitations, pauses and very slow speech; Repetitions on /u-un/ one) /t/ (trois/three), /q/ quatre (four). It is clear that counting is very effortful (even counting 1-3),
- Reciting the days of the week: same loss of automatic series, hesitations and abnormal pauses were present.
- Reading aloud (3 times the same text) revealed no adaptation effect that is normally present in developmental stuttering, repetitions and blocks, inability to tell the simple content of the story, and great fatigue after reading.

- Repeating of words, sentences, proverbs showed the same difficulties as in reading
- In his spontaneous speech and dialogue the same repetitions, blocks, and abnormal pauses were present

Fluency-enhancing conditions

During whispered speech, there was no stuttering observed. The same fluent speech was manifest during speech with prolongation of the first syllable.

But:

Stuttering was present during choral reading; the patient was unable to follow a rhythm and to anticipate a regular rhythm /beat of the metronome. There was no adaptation effect in choral reading and speaking

4. The need for a differential diagnosis

As a result a complex and contradictory picture of his stuttering and other complaints was noted.

In order to make a differential diagnosis, several hypotheses were presented to be analyzed:

a. PTSD and psychogenic stuttering?
b. PTSD and aphasia and stuttering?
c. PTSD and drug-induced stuttering?
d. PTSD and stuttering are linked syndromes?
e. Stuttering due to Neurobiological alterations in PTSD
f. PTSD and Stuttering due to traumatic brain Injury (TBI)?

4.1. PTSD and psychogenic stuttering

Deal (1982) and Rentschler (1984) relate psychogenic stuttering of sudden onset following a traumatic incident, as associated with lack of adaptation and stuttering in automatic series. According to these authors, psychogenic stuttering with neurogenic-like symptoms of sub-cortical origin (dysfunction of the basal ganglia circuit) can be caused by stress and is often accompanied by a state of confusion. But: We have seen in the clinic that psychogenic stuttering is generally resolved when the causes have been diagnosed and treated. In the case of this patient, the probable cause was clearly identified (the accident) but stuttering continued and worsened….Therefore the second hypothesis was tested:

4.2. PTSD, aphasia and concomitant stuttering

The patient showed aphasic symptoms, such as: word-finding problems, difficulty in remembering words/numbers, difficulty in remembering a just-read text, difficulty in copying a block design. But: word finding problems resolved during the following months although the difficulties in remembering a just read text and the stuttering were still present.

4.3. PTSD and Drug induced stuttering

The patient could not tolerate the following drugs: Mirtazapin, Remergon, Seroxat. Drug-induced stuttering is documented after medication with clozapine, olanzapine, risperidone, haloperidol, trifluoperazine (Brady, 1998). On the other hand, seizures and stuttering have been reported with high doses of clozapine (200-250 mg/day), (Duggal et al., 2002). Krishnakanth et al. (2008) in their study came to the conclusion that generally speaking, stuttering disappears after stopping or reducing the drug. But: the patient stopped using all drugs but the stuttering didn't disappear and is still present (2 years later).

4.4. PTSD and Stuttering are linked syndromes?

The findings of Shin (2006), Sherin et al. (2011), Bremner (2008), Wu et al. (1997) and Lu et al. (2010) about PTSD and neuro-biological alterations after psychological trauma reflect earlier research on stuttering and basal ganglia implications. As a matter of fact, in 1976 Lamendella stated that the emotional language was the expression of the Limbic System through the Basal Ganglia and the Limbic System.

Baxter et al. (1992) observed lesions in the Anterior Cingulate that gave rise to emotional utterances. Freire Maia et al. (1999) noted a strong interaction between Basal Ganglia and Limbic System (RMI studies) in the expression of emotional utterances. Wu et al. (1997) and Lu et al. (2010) came to the conclusion that altered connectivity and anomalous anatomy in the basal ganglia circuit were present in the scans of stutterers. De Nil et al. (2001, 2003) noted higher activity in ACC in stuttering persons compared to non-stuttering people. Alm (2004) wrote a comprehensive review of the literature on Stuttering and Basal Ganglia. Could it then be that PTSD and stuttering are not only linked, but follow the same process?

Starkweather and Givens (2004) developed a theory of an identical process of PTSD and stuttering, with patterns of dissociation, avoidance, repetitive experience of fear and hyper arousal associated with PTSD and stuttering. But if this is so, stuttering is then a very specific form of PTSD. But in this case:

- How can we explain that PTSD is rarely accompanied by stuttering?
- How can we explain the differences in functioning?
- Does the chronic stutterer have the same daily life problems as the PTSD patient (nightmares, fatigue flashbacks, sleeping, memory, concentration?)
- Is the stutterer unable to work and to function like a PTSD-patient?
- Is dissociation a form of psychological necessity of survival or a result of neurological disruption?
- Is dissociation present in most stutterers?

If there are elements of convergence or resemblance between PTSD and Stuttering, due to stress and basal ganglia dysfunction, there is a difference in degree and in every day life experience of the trauma (fear) and difference in risk factors.

4.5. Stuttering due to Neurobiological alterations in PTSD

There are several studies on neurobiological alterations in PTSD, such as: endocrine dysregulation from the hypothalamic-pituitary-adrenal axis/loop (cortisol release, adrenal hormones: response to stress) and neuro-chemical dysregulation through neurotransmitters (catecholamine, serotonin and dopamine) on hippocampus, hypothalamus, thalamus and amygdala in PTSD (Sherin et al., 2011). Changes in brain functioning in PTSD can include sub cortical and cortical dysfunction of hippocampus, amygdala, thalamus; loss of inhibitory function of anterior cingulate and orbito frontal region; dysfunction of the basal ganglia circuit (Bremner et al., 2003, 2008), van Reekum et al., 2000). Reduced hippocampal volume and hyper arousal of amygdala was found in the studies by Schwarzbold et al. 2008 and van Reekum et al., 2000.

Bremner (2008) observed in his patients dysfunction of the medial prefrontal cortex (m PFC) and Anterior Cingulate Cortex (ACC) that caused failure of extinction of fear due to glutamatergic alterations. Other dysfunctions of the basal ganglia due to stress were found in the study of Sherin et al. (2011); and in the very complete review by Alm (2004).

4.6. TBI and PTSD and Stuttering

The 1st scan (11/05/2011), revealed a serious enlargement of ventricular system; hypo-density in white matter structures, non-specific leuco-encephalopathy in fronto-parietal region, but did not detect post-traumatic lesions. It is known that mild TBI and PTSD are associated with mild axonal injury in brainstem, frontal, prefrontal and temporal regions, with sub-cortical involvement (hypothalamus, amygdala, BG and thalamus); injury of the cortico-striatal-pallidal-thalamic pathway that plays a role in the manifestation of emotions, stress, learning and stuttering. At the same time, high cortisol release diminishes the axonal endings of the hippocampus, which as a result causes the inhibition of hippocampal neurogenesis and gives rise to learning and memory disabilities as shown by the studies of Sherin et al. (2011), Tanaka et al. (1997), Bremner (2008). Those disabilities can be manifest some weeks after TBI.

Reduced volume of hippocampus and hyper arousal of amygdala in PTSD patients were also observed by Schwarzbold et al. 2008; van Reekum et al. 2000. According to Mayou et al. (1993), TBI and PTSD are associated with changes in mood, personality and behavior, and emotional instability. On the other hand, PTSD is not associated with a neurotic predisposition, but is strongly associated with horrific memories of the accident. The authors observe that PTSD does generally not occur in patients who lost consciousness.

4.7. How to understand all these elements in the case of our patient?

The clinical situation of JP reveals very slow speech and stuttering, no adaptation in reading, abnormal pauses, loss of automatic series, memory deficits, total incapacity to work, nightmares and flashbacks, depressive state with loss of libido; fatigability, slight insensibility of right hand, leg and face, high emotional arousal and instability.

Neurolinguistic observations reveal dissociation between excellent receptive, semantic comprehension and very bad oral production, slight deficit of short term memory, deficit of working memory, normal long term memory, severely delayed reaction time.

All these observations point to a possible TBI and that is the reason of a new PET scan and f MRI investigation (09/03/2012).

4.8. PET scan results

- Scan with fluorodeoxyglucose:
- Ventricles: highly increased volume
- Low activation of left temporal and parietal associative cortices (hypo metabolism)
- Metabolic deficit of the left thalamus
- Lesions of the lateral medial frontal and orbital frontal regions
- Multi-focal pathology with repercussion on the left thalamus consistent with multiple axonal lesions, independent of the observed global ventricular dilatation

4.9. Results of fMRI

- Working memory: activation of fronto-temporal and parietal regions, left side more than right;
- Very high activity of the cerebellar vermis compared to controls (cf. language problems)
- Repetition of words and sentences: normal activation of Broca, frontal and pre motoric regions
- Abnormal ventricular volume
- Hypo density of the white matter

4.10. General conclusion based on the fMRI

Hypometabolism of the left temporal, parietal, fronto-orbital regions and thalamus, compatible with diffuse axonal lesions and working memory deficits, with a progressive character (Majérus & Salmon 2013).

5. Discussion

fMRI revealed volume loss of left thalamus which is compatible with language problems. Indeed, early studies in the seventies with deep electric stimulations of left anterior thalamus provoked stuttered speech (Andy & Bhatnagar, (1989, 1992), Ojemann (1976) and Schaltenbrand (1975). Other studies by Cipolotti (1988), Lebrun et al. (1990) showed that lesions of the left thalamus can provoke neurogenic stuttering. Thus, the observations of the f RMI are consistent with previous studies and observations of neurogenic stuttering and PTSD. The psychiatric diagnosis of PTSD and stuttering due to shock/ stress and the observation of leuco-encephalopathy ante-trauma with a slow evolution over time are not confirmed by the facts (no slow evolution but a brutal change in neural functioning... the day after accident)

As discussed before in this paper, mild TBI can give rise to axonal lesions and neurologic cortical and sub-cortical deficits, loss of memory, and slow speech; those neurologic deficits can worsen over time (cf. stuttering onset a month after accident, but slow speech the day after the accident). Lesions in the Basal Ganglia can give rise to stuttering and motor problems as shown in several studies mentioned above and is consistent with stuttering research. As to the patient, his family could certify that stuttering had never been present before the day of the accident. The multilingual patient had a position of responsibility in an international business and had been performing successfully during 35 years. Until the day of his accident he had no complaints of any sort (no headache, fatigue, loss of concentration, insensibility in the right arm or leg) that could refer to existing psychiatric or neurologic deficits or leuco-pathology. He had no psychiatric antecedents.

6. Conclusion

The neurolinguistic approach of the stuttering symptoms together with the observation of the general slow functioning and the typical symptoms of PTSD of this patient pointed to PTSD with neurologic injury that could explain the stuttering, the slow speech, the general slow functioning, the loss of memory and his inability to work normally. This neurolinguistic observation and diagnosis of TBI, the neurogenic stuttering and the neurologic deficits were confirmed by he neurologists as shown by their analysis of a PET scan and f RMI.

Thus, PTSD, TBI and Stuttering can have some amazingly similar symptoms and neurobiological impact, justifying the differential diagnosis and the need for more research.

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